AMENDMENT IN THE CLAIMS:

Kindly amend the claims, without prejudice, without admission, without surrender of subject matter, and without any intention of creating any estoppel as to equivalents, to read as follows:

1. (Currently amended) A transgenic mouse model showing hypomyelinosis of the thalamus that can be a cause of Nasu-Hakola disease, and showing a neuropsychiatric disorder caused by the hypomyelinosis, wherein the transgenic mouse comprises a homozygous disruption in chromosomal DAP12 (DNAX Activation Protein 12) gene function, and wherein the homozygous disruption includes the promoter region and exons 1, 2, and 3.

2. (Canceled)

3. (Currently amended) The transgenic mouse model of claim 1, wherein the homozygous disruption in DAP12 can be pheonotypically exhibited as a myelinogenesis developmental disorder ~~or a neuropsychiatric disorder associated with disruption in DAP12 gene function~~ that can be a cause of Nasu-Hakola disease.

4.(Currently amended) The transgenic mouse model of claim [[3]] 1, wherein the neuropsychiatric disorder caused by hypomyelinosis is selected from the group consisting of Nasu-Hakola disease caused by hypomyelinosis, dementia ~~associated with disruption in DAP12 gene function~~ caused by hypomyelinosis, schizophrenia ~~associated with disruption in DAP12 gene function~~ caused by hypomyelinosis, schizotypal personality disorders ~~associated with disruption in DAP12 gene function~~ caused by hypomyelinosis, obsessive-compulsive disorders ~~associated with disruption in DAP12 gene function~~ caused by hypomyelinosis, or Tourette's syndrome ~~associated with disruption in DAP12 gene function~~ caused by hypomyelinosis.

5. (Currently amended) The transgenic mouse model of claim [[3]] 1, wherein the neuropsychiatric disorder caused by hypomyelinosis is Nasu-Hakola disease caused by hypomyelinosis or dementia ~~associated with disruption in DAP12 gene function~~ caused by hypomyelinosis.

6-18. (Canceled)

19. (Previously presented) The transgenic mouse model of claim 1, wherein the expression of myelin basic protein in the brain is weak in regions where DAP12 is strongly expressed in wild-type mice.

20. (Previously presented) The transgenic mouse model of claim 1, wherein the transgenic mouse exhibits an impairment in sensorimotor gating as compared to wild-type mice.